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ON THE POSSIBILITY OF THE OCCURRENCE OF TRYPANOSOMIASIS IN INDIA

C. DONOVAN

British Medical Journal, 1903, ii.

22200058546

infection is the enlargement of the adjacent lymphatic glands; and here enters a great problem, the part played by the lymphatics in arresting tuberculosis. The enlargement of the glands, although in a sense evidence of disease, is at the same time the outcome of a battle fought to resist disease. The lymphatics form not only the lines of defence, but also, where inefficient, the paths of general invasion, and we have to consider this when we come to deal surgically with a case of primary infection.

In secondary infection of the ear with tubercle there is comparatively, if not entirely, an absence of enlargement of the adjacent lymphatics. Why they are not affected in the secondary form I must leave for a paper by itself.

A second point in differential diagnosis between primary and secondary infection is that in the latter there is evidence of tubercle elsewhere, commonly in the lungs, and, as I have already said, with cavity formation. The tonsils and adenoids, when they play a part in the infection, I regard rather in the light of half-way houses than as primary foci.

A third point is the presence or absence of intracranial complications. It is hardly correct, without a qualifying clause, to say that tuberculous suppuration leads much less often than other forms of middle-ear suppuration to intracranial complications. Intracranial complications seldom occur in the secondary form, but in the primary form the child is not uncommonly killed by a meningitis, as part of a general blood infection.

The practical bearing which discrimination between primary and secondary infection has upon the surgical treatment—and by that I mean an attempt to eradicate the bone disease—may be briefly stated as follows:

In the primary form, when the adjacent glands are involved, let these be removed, first attacking the more distal and least affected, the intention being to cut off the spread of infection, and so localize the focus of disease to be removed at a later date.

When the disease is secondary to advanced pulmonary tuberculosis, a mastoid operation is seldom required; on the contrary, a mastoid operation is more likely to be what a tracheotomy is to laryngeal tuberculosis—the beginning of the end.

I am desirous of expressing my indebtedness and thanks to the British Medical Association, under whose auspices the research, of which this is the record of a part, is being conducted.

REFERENCE.

[1] The Morphological and Physiological Variations of the Bacillus Tuberculosis and its Relations. By William Bulloch, M.D. *Transactions* of the British Congress on Tuberculosis, vol. iii, p. 494.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

ON THE POSSIBILITY OF THE OCCURRENCE OF TRYPANOSOMIASIS IN INDIA.

WITH regard to Major Leishman's contribution under the above head in the BRITISH MEDICAL JOURNAL of May 30th, I wish to state briefly that I have noted bodies similar to those described by him in smears taken *post mortem* from enlarged spleens of patients—natives of India—said to have died of chronic malaria. I obtained them in three consecutive cases on April 9th, 23rd, and 24th, 1903.

In the first instance, I thought I had discovered the long-sought-for resting-stage form of the malarial parasite in man, but could not compare them with any analogous stages in the sporozoa. However, on again procuring the same bodies in the two other cases, I changed my views, and considered they were probably *post-mortem* degenerations of the nuclei of the splenic pulp cells.

On reading Major Leishman's paper, I at once recognized the similarity of his so-called degenerations of the trypanosomata to those found by me in the spleens of the cadavers above mentioned.

Yesterday (June 17th) I had occasion to puncture *intra vitam* the spleen of a native boy, aged 12 years, suffering from irregular pyrexia, with no malarial parasite in his peripheral blood (careful examination of stained films on four several occasions), and found identical bodies in the blood from the spleen, thus removing any doubt there was as to the products being due to *post-mortem* changes.

It is unwise to theorize on the insufficient grounds at present in hand. I hope to contribute something more definite on the subject after further and more prolonged study of these organisms. My films were stained by the Maurer-Romanowsky method. I am familiar with the appearance of the trypanosomata—T. evansi, T. lewisi, and a species doubtful occurring in the blood of the common Indian squirrel (Scirrus palmarum). There was nothing resembling trypanosomata in the peripheral blood of the boy in question.

C. DONOVAN, Captain I.M.S.,
Second Physician, Government General Hospital, Madras.

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A CASE OF ASCITES MOST PROBABLY CAUSED BY INFECTIVE THROMBOSIS OF THE HEPATIC VEINS.

DURING 1902 I visited five times in consultation with her medical man a female patient, aged 47. She was well nourished, and of very healthy appearance. She was married, had had two children, the youngest aged 11 years, and two miscarriages since, the last one seven years ago. Her family history was good.

My first visit was March 3rd, 1902, when I was told that she had been in bed for two months before Christmas with cellulitis of one arm. This was dealt with by an operating surgeon, and 14 incisions were made, but no pus came out. The surgeon thought the cause was partly lymphangitis and partly thrombosis. The arm was completely restored. The patient said that she had had white leg at least once after a confinement.

At Christmas there was a sense of fullness in the abdomen. She had not menstruated since the middle of October, having been quite regular up till then. Ascites was recognized a week or so before my first visit, and was attended with pain and tenderness in both iliac regions. The ascites increased, and began to interfere with the breathing; there was also some slight swelling of the ankles. I could not feel the liver or spleen, and there were no superficial abdominal veins to be seen. By a process of exclusion I narrowed down my diagnosis to infective thrombosis of the hepatic veins. There was no likelihood at all of syphilis notwithstanding the miscarriages, although there had been a tendency to thrombosis of other veins in former illnesses. More than once we thought we must recommend paracentesis, but by rest and elaterium, which she stood well, and by inducing profuse diaphoresis and diuresis the patient after three or four months made a complete recovery. It is possible that the climacteric was intimately connected with the *fons et origo mali*.

HENRY WALDO, M.D., M.R.C.P.,
Physician to the Bristol Royal Infirmary.

CASE OF PROLAPSUS UTERI TREATED BY PARAFFIN INJECTIONS.

THE patient, aged 69, and too feeble for a radical operation, had for the last twelve years suffered from complete prolapsus uteri. The uterus was nearly always down, even when she was in bed, and was a source of much misery. The perineum was rather less than an inch in length. Pessaries had been tried but failed to keep the prolapse up.

Operation.—May 24th, 1903. The patient was anaesthetized and placed in the lithotomy position. The method followed was that suggested by Mr. Stephen Paget (*Lancet*, May 16th, 1903). Paraffin melting at about 46° C. (115° F.) was used, and injections of about 9 c.cm. (f. ʒ iiss) each were made in several places under the mucous membrane of the lateral and posterior vaginal walls. Two or three injections were also made about the cervix. About 95 c.cm. (f. ʒ iij ʒ iij) of paraffin were used in all. A finger was kept in the rectum to guide the posterior and lateral injections: and a probe was passed into the urethra while making the more anterior injections. An ordinary antitoxin syringe was found to act well. The operation lasted about one hour and a quarter, some time being lost by the paraffin setting in the needle of the syringe.

To speak of cure at present would be, perhaps, premature, but the result has been surprisingly satisfactory. The lumen of the vagina has been very much narrowed and its walls stiffened. The uterus is now completely held up during

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ON THE POSSIBILITY OF THE OCCURRENCE OF TRYPANOSOMIASIS IN INDIA.

With regard to Major Leishman's contribution under the above head in the British Medical Journal of May 20th, I wish to state briefly that I have noted bodies similar to those described by him in smears taken post mortem from enlarged spleens of patients—natives of India—said to have died of pernicious malaria. I obtained them in three consecutive cases on April 6th, 23rd, and 24th, 1903.

In the first instance I thought I had discovered the long-sought-for resting-stage form of the malarial parasite in man, but could not compare them with any analogous stages in the quotidian or tertian forms of the parasite. However, on again meeting the same bodies in the two other cases, I changed my views, and considered they were probably post-mortem degenerations of the motile forms of the aplenic pulp cells.

On reading Major Leishman's paper, I at once recognized the similarity of his so-called flagellate stages of the trypanosomata to the bodies found by me in the cadaveric spleens mentioned.

Therefore (since 1903 I had occasion to perform several post-mortem examinations, suffering from malarial cachexia, with a view to ascertaining if the parasites were present in my peripheral blood, but in no case did I find them. Nor did I succeed in finding them in the blood from the spleen, though they could always be found there in the pernicious cases. I am unable to offer any explanation of the absence of the parasites in the peripheral blood, being due to post-mortem changes.

It is useless to theorize on the significant granula at present in them. I hope to contribute something more definite on the subject after further and more prolonged study of these organisms. My films were stained by the Romanowsky-Nocht method. I am familiar with the appearances of the trypanosomata—T. evansi, T. lewisi, and a species doubtful occurring in the blood of the common Indian squirrel (Sciurus palmarum). There was nothing resembling trypanosomata in the peripheral blood of the boy in question.

G. DESOUZA, Captain I.M.S.,
Assistant Professor, Government General Hospital, Madras.

A CASE OF PARALYSIS MOST PROBABLY DUE TO INJECTIVE THROMBOSIS OF THE SUBCLAVIAN VESSELS.

[illegible] 1902 I visited five times in consultation with Dr. [illegible] a female patient, aged 42. She was married, had had [illegible] very healthy appearance. She was married, had had two children, the youngest aged 11 years, and two miscarriages since, the last one seven years ago. Her family history was good.

My first visit was March 3rd, 1902, when I was told that she had been in bed for two months before. Christmas with cellulitis of the arm. This was dealt with by an operating surgeon, and 14 stitches were made, but no pus came out. The surgeon thought the cellulitis was partly lymphangitis and partly thrombosis. The arm was completely relieved. The patient said that she had had trouble for at least one year a continuance.

At Christmas there was a series of failures in the abdomen. She had not been natural since the middle of October, having been quite regular up till then. Aperies was noticed a week or so before my first visit, and was attended with pain and tenderness in both iliac regions. The aperies increased, and began to observe with the diarrhœa; there was also some slight swelling of the ankles. I could not feel the liver or spleen, and there were no superficial abdominal veins to be seen. By a process of exclusion I reached the conclusion that the diagnosis to infective thrombosis of the pelvic veins. There was no likelihood at all of syphilis notwithstanding the previous cellulitis, although there had been a tendency to thrombosis in other veins in former illnesses. More than once we thought we must recommend potassium iodide, but by recollection of what she stood well, and by the inducing positive diagnosis and diagnosis the patient after three or four months passed complete recovery. It is possible that the climacteric was ultimately connected with the case as it arose such.

HENRY HARRIS, M.D., M.R.C.P.,
Physician to the Mental Hospital, Dublin.

CASE OF TROPICAL ULCER TREATED BY PARAFFIN INJECTIONS.

The patient, aged 60, and too feeble for a radical operation, had for the last twelve years suffered from complete paralysis of the bladder. The uterus was nearly always down, even when she was in bed, and was a source of much misery. The previous ulcer was rather less than an inch in length. Pressure had been tried but failed to keep the prolapse in.

On 23rd May, 1903. The patient was anæsthetized and placed in the lithotomy position. The method followed was that suggested on July, Stephen Paget (Lancet, May 16th, 1903). Paraffin melting at about 45° C. (113° F.) was used, and injections of about 6 c.cm. (1 3/5 ths) each were made in several places under the mucous membrane of the lateral and posterior vaginal walls. Two or three injections were also made about the cervix. About 95 c.cm. (1 3/5th) of paraffin were used in all. A finger was kept in the rectum to guide the posterior and lateral injections; and a probe was passed into the uterus while making the anterior injections. An ordinary hypodermic syringe was found to act well. The operation lasted about one hour and a quarter, some time being lost by the paraffin setting in the needle of the syringe.

To speak of cure at present would be, perhaps, premature, but the result has been surprisingly satisfactory. The lining of the vagina has been very much improved and its walls stiffened. The uterus is now completely held up during